

CASE REPORT

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Spontaneous Splenic Rupture Following Intravenous Thrombolysis with Alteplase Applied as Stroke Therapy – Case Report and Review of Literature

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ABSTRACT

Introduction: Stroke is a medical emergency in neurology, and is one of the leading causes of death nowadays. At a recent time, a therapeutic method used in adequate conditions is thrombolysis, a treatment of an emerging clot in the brain vascular system by alteplase. The application of alteplase also has a high risk of life threatening conditions. **Case report:** This is a brief report of a case with thrombolysis complication which manifested as a spleen rupture.

Key words: Stroke; Thrombolysis; Spleen rupture.

1. INTRODUCTION

Despite significant improvement in therapy in the past two decades, stroke is the leading cause of death and disability worldwide (1). The use of recombinant tissue Plasminogen Activator (IV-tPA) is a well-established standard of treatment for patients presenting with acute ischemic stroke (AIS) within the first 4.5 h from the symptom onset (2-5). In order to minimize the unwanted side effects (6), there are clear and strictly defined criteria for appropriate patient selection for the systemic treatment with rt-PA. Intravenous application of alteplase is the only therapy approved by the US Food and Drug Administration (FDA) for the treatment of patients with ischemic stroke. Its use is associated with improved outcomes for a wide group of patients (7) and its earlier treatment is associated with better outcomes (8).

During thrombolytic therapy, there is a risk of intracerebral hemorrhage with a reported incidence of about 6% (9, 10). Other, less common complications of thrombolytic therapy include systemic hemorrhage, angioedema and allergic reactions (11).

We report a case of a female patient with AIS who developed symptoms of internal bleeding shortly after administration of thrombolytic therapy. A diagnosis of spontaneous splenic rupture was made by the following clinical and radiological examination.

2. CASE REPORT

A 45-year old female patient was admitted to hospital with weakness of the left extremities, dizziness, nausea and vomiting. In the past medical history, she asserted frequent urinary infections and hyperventilation as a result of panic attacks, and therefore she was tak-

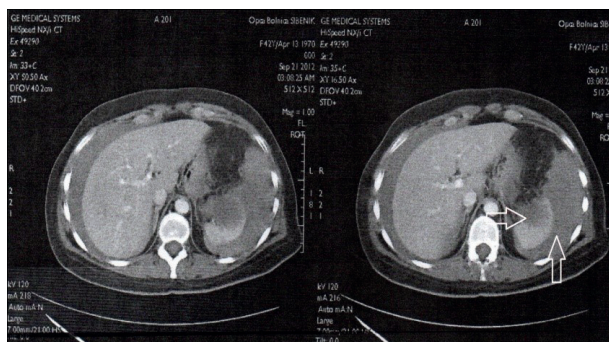


Figure 1. CT scan of abdomen and pelvis showed a splenic rupture with massive intraperitoneal bleeding in the area of ruptured spleen, perihepatic space, in both paracolic gutters and in the pelvis

ing oxazepam temporarily. On admission, dysarthria, eyeballs deviation to the right, moderate paresis of the left hand, plegia of the left leg, diminished ipsilateral reflexes, and diminished left plantar reflex were observed in the neurological status. She was hemodynamically stable with blood pressure of 110/70 mmHg. The National Institutes of Health Stroke Scale (NIHSS) was 12. Computed tomography (CT) of the brain was normal on admission. Laboratory findings showed increased blood glucose level (7.4 mmol/l) and low potassium level (3.5 mmol/l). Other parameters were within their reference values. The patient satisfied the criteria for thrombolytic therapy and intravenous alteplase was administered at the dose of 0.9 mg/kg. Two hours after the thrombolysis, chest pain, headache and vomiting occurred. An electrocardiogram (ECG) showed no signs of acute ischemia. Mild dysarthria, incomplete left homonymous hemianopsia, left-sided central facial palsy, mild to moderate paresis of the left hand, severe paresis of the left leg and left hemihypesthesia were observed in neurological status. NIHSS score was 11. Control CT of the brain performed six hours after admission showed no signs of intracerebral hemorrhage or ischemia. The patient developed tachycardia with a rate of 125 bpm, her blood pressure dropped to 75/55 mmHg and hemoglobin level decreased from 139 g/l to 99g/l, which indicated the bleeding occurrence and patient was moved to the intensive care unit. An urgent CT scan of abdomen and pelvis was performed and it showed a splenic rupture with massive intraperitoneal bleeding (Figure 1). The patient underwent splenectomy. On control neurological examination, left homonymous hemianopsia, unreactive left pupil, moderate paresis of the left hand, plegia of the left leg, heightened reflexes and positive Babinski sign left were observed. The patient's speech was normal. Control CT scan of the brain performed a week after admission showed demarcation of an extensive hypodense lesion in the irrigation area of the right posterior cerebral artery. An abdominal ultrasound performed before discharge was normal. The patient was discharged with a recommendation for physical therapy, appropriate for her condition.

3. REVIEW OF LITERATURE

Spleen rupture is a rare but life-threatening complication of thrombolytic therapy. Only a few cases have been described to date. In two cases, there was a history of significant trauma prior to thrombolysis (12, 13). Friedrich and colleagues described a case of a patient with polycythemia rubra vera in which spontaneous spleen rupture followed thrombolytic therapy for myocardial infarction (14), while in the case described by Jankowski et al, spontaneous spleen rupture after application of thrombolytic therapy for AIS was preceded by splenomegaly secondary to infection (15). A few cases in which spleen rupture followed the streptokinase therapy have been reported (16-19). In one of these cases, thrombolytic therapy was used for deep venous thrombosis (16), while in other cases it was used for acute myocardial infarction (17-19). In a case described by Cheung and colleagues, 3 hours after administration of the t-PA for acute myocardial infarction, shock and acute abdomen developed due to spleen rupture (20). Revesz et al reported a case in which spontaneous spleen rupture followed t-PA for peripheral arterial occlusion (21). Harries and Gomez reported a case of a patient with acute myocardial infarction, in which spleen rupture occurred as a complication of the thrombolytic therapy with tenecteplase (22). Spontaneous spleen rupture following alteplase therapy for stroke was reported only once (23).

4. DISCUSSION

The aim of current therapy for AIS is to improve the patients' long-term functional outcome. To date, the only proven therapy for AIS is early recanalization (24). The FDA approved the use of alteplase in treatment of ischemic stroke based on the results of the NINDS (National Institute of Neurological Disorders and Stroke) study in 1996 (2). In this study, the time window was 3 hours and it showed that alteplase-treated patients had a 30 % higher probability of recovering with little or no deficit after 3 months. This clinical benefit of alteplase treatment overcame the risk of symptomatic intracerebral hemorrhage that occurred in 5.8% of cases, compared with placebo in which intracerebral hemorrhage was reported in 0.6% of cases (26). Several years later therapeutic time window was extended up to 4.5 hours, based on results of the ECASS III (European Cooperative Acute Stroke Study) study (26). However, this study excluded patients over 80 years of age, those with severe stroke (clinically with NIHSS > 25, or radiologically with more than one-third of the middle cerebral artery territory involved) and patients with a combination of diabetes and a prior stroke.

Alteplase promotes thrombolysis by hydrolyzing the arginine-valine peptide bond in plasminogen to form the active proteolytic enzyme plasmin, leading to an effective fibrin clot dissolution. In contrast to streptokinase and urokinase, alteplase is relatively fibrin-selective plasminogen activator, and therefore it is relatively inactive in the systemic circulation (27). Beside the risk of intracranial hemorrhage and allergic reactions as adverse effects of alteplase, note should be taken of angioedema

(11), that occurs in 1-2 % of cases and it is more common in patients taking ACE inhibitors) (28).

The benefit of thrombolytic therapy is undeniable, but each patient receiving it should be observed carefully. Although rare, splenic rupture should be considered in any patient receiving thrombolytic therapy, with signs of systemic bleeding, especially in those in which there is a history of trauma or of spleen disease, which is a predisposing factor for rupture.

In our case, there was no history of trauma, myeloproliferative diseases or splenomegaly. Signs of systemic bleeding developed shortly after administration of alteplase and CT scan of abdomen and pelvis confirmed the splenic rupture.

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